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SPECIAL SECTION

Pluralism and Heuristic Identification

Some Explorations in Behavioral Genetics

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ABSTRACT. In this paper, two recent interpretations of current work in behavioral genetics are rejected. Genetic reductionism, the view according to which genetic properties are causally sufficient for phenotypic traits, is dismissed because it ignores the fact that there are not only causal pathways from DNA to phenotype but also pathways that feed back from, for instance, the cytoplasm or the outside world to the genetic material. The complexity of development is acknowledged in the view known as developmentalism, which claims that a complex causal network of genetic and non-genetic factors is responsible for phenotypic outcomes. However, genetic explanations, at least in some contexts, do seem to have a privileged status. Heuristic identity theory appears to offer a more adequate interpretation for behavioral genetics. It is in many cases very illuminating to hypothetically identify phenotypic traits with genetic properties. We claim that McCauley and Bechtel's proposal calls for at least two constraints to avoid a wild proliferation of implausible identities. First, more emphasis should be placed on explanatory *failures*. Second, the most interesting identifications are those that make 'qualitative leaps', that is, they must apply across distinct levels of analysis.

KEY WORDS: behavioral genetics, heuristic identity theory, phenotypic outcomes, reductionism

The Problem of Genetic Reductionism

In the contemporary popular press we can read on a regular basis about the discovery of 'genes for' certain phenotypic traits. For instance, it has been suggested that we now know the genes for alcoholism, agression, homosexuality, sensation seeking, depression, intelligence, learning and schizophrenia (see, e.g., Hamer & Copeland, 1998), and even for voting, military combat and watching TV (mentioned in Rose, 1995, p. 647). Although it must be emphasized that publications in which genetic discoveries are reported are often much more careful in their phrasing than are newspaper and magazine

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articles, talk of 'genes for' may still easily slide into simple-minded forms of genetic reductionism.

In its classical form, behavioral genetics is population-based; it attempts to assess the genetic contribution to phenotypic differences. The traditional methods of behavioral genetics (i.e. twin, adoption and family studies) allowed it to partition the population variance into genetic and environmental portions (see, e.g., Bouchard, 1994; Bouchard, Lykken, McGue, Segal, & Tellegen, 1990; Plomin, DeFries, McClearn, & Rutter, 1997; Wright, 1999). The goal was to estimate the genetic contribution, describing genetic differences within populations, in the statistical notion of the heritability of traits. After its recent confluence with molecular biology, a number of new techniques have allowed behavioral geneticists to go hunting for real genes. These methods give the impression of being capable of building bridges between quantitative and molecular genetics. On the basis of the heritability coefficient of classical behavioral genetics, no inferences could be made with respect to the nature of the causal path from genotype to phenotype. With these molecular techniques, this appears to be a real possibility (Wahlsten, 1999; Wolf, 1995). Thus, genuine reduction, not just population statistics, enters the picture.

For instance, with so-called 'QTL (*quantitative trait loci*) analysis', quantitative behavioral genetics has found a way to move beyond heritability to identify individual genes and gene complexes and to reveal the nature of genotype/phenotype relations. What is special about QTL analysis is that it is able to deal with interacting arrays of genes. A QTL refers to multiple chromosomal loci that together contribute to the variance of a trait. Typically, the experimental design behind QTL analysis is exploited to pin down genetic influences with varying (and often small) effect sizes. Leaving aside the niceties, QTL identification may be effected through a number of approaches, for instance linkage analysis and allelic association (Plomin et al., 1997, pp. 94–95). The question is whether this new kind of behavioral genetics may avoid interpretations in terms of genetic reductionism. According to *genetic reductionism* in its most simplistic, preformationist form, genetic properties are causally sufficient for phenotypic traits.

At this point it should be noted that genetic reductionism in that sense is not underwritten by the received view of theory reduction as inspired by Ernest Nagel (1961), requiring the formulation of bridge laws between different theories or sciences. Most reductionists who base themselves on this classical model have suggested that identities should be established between different properties of the world. The textbook example here is the formulation of a bridge law between thermodynamics and statistical mechanics that brings out the (ontological) identification of temperature and mean kinetic energy of molecules. However, in the case of behavioral genetics such strict identities are absent, as genotypes and phenotypes enter into causal relationships with one another, which means that they cannot be

ontologically identified (in any strict sense of the term). The interpretation that is usually given to genetic reductionism, then, is one in terms of 'causal sufficiency'. A reduction of phenotype to genotype takes place when the latter is causally sufficient for the former. However, as will become apparent at a later stage of this paper, in a way it may still be fruitful to speak of the establishment of identifications (albeit in an attenuated sense) between genes and phenotypic traits. Basically, the idea is that in the behavioral genetics case one searches for (heuristic) identifications of functions and causal mechanisms.

Not all biologists will subscribe to the 'causal sufficiency' type of reductionism (as they will concede that a cytological environment will be causally required as well), although probably many will be willing to defend more refined forms. The popularity of metaphors like 'genetic program', 'genetic blueprint' and 'mastermolecule' (Oyama, 1985; Van der Weele, 1999) indicates that many biologists and philosophers of biology do assign causal and informational priority to the genome. Rosenberg (1994), for instance, subscribes to the view that '[t]he information that controls development and heredity for all organisms is carried in nucleic acid and only nucleic acid' (p. 27). The genome is like a program; it is a 'set of instruction books' (Watson, 1990, p. 44; cf. Mayr, 1982, p. 824) on the basis of which development, cognitive capacities and behavior are tightly and algorithmically controlled. This implies a kind of *computational reductionism*: the embryo can presumably be computed from a full specification of the DNA in the egg (Rosenberg, 1997).

The idea of genetic control is clearly expressed in Francis Crick's *Central Dogma* of molecular biology. In 1957, Francis Crick, with James Watson Nobel laureate for the celebrated 1953 discovery of the double-helix structure of DNA, formulated this dogma, which states that there is a one-way flow of information from DNA via RNA to proteins. This process of gene expression takes roughly two steps. First, messenger RNA (mRNA) is *transcribed* from DNA templates. Second, this mRNA travels from the nucleus of the cell to the cytoplasm, where it is *translated* into the amino acid sequences that constitute a protein. There is a one-to-one mapping between the nucleotide sequences in the DNA and in the mRNA. Similarly, there roughly exists a one-to-one correspondence between the nucleotide sequence in the mRNA and the order of amino acids in the protein that is coded for by the genome.

According to Crick's 'DNA $\rightarrow$ RNA $\rightarrow$ protein' dogma, genes control development from the zygote to the adult state (see, e.g., Walbot & Holder, 1987, p. 4; Wolpert, 1998, pp. 13–15). The flow of information is unidirectional. This results in reductionism: if one knows the entire DNA sequence, one could, according to the Dogma, in principle (though perhaps not in practice due to epistemic limitations) *deduce* protein structure from a specification of the genetic code. Moreover, the Central Dogma did much to

secure the marginalization of developmental biology with respect to evolutionary biology, and to isolate ontogeny from phylogeny (Depew & Weber, 1995, p. 396). As Watson (1993) has recently stated: 'Now developmental biologists, who do not think in terms of DNA, are relics of the past with little likelihood to influence the future' (p. 313).

Some theorists have claimed, however, that the Central Dogma is inadequate to capture the complexity and richness of organismic development. There are a number of reasons to doubt the validity of the Central Dogma. One is that certain gene products are able to activate and inactivate genes (Gottlieb, 1992, p. 140). Additionally, even states of the outside world are known to influence gene expression and development directly (e.g. temperature in the environment determines sex expression in Mississippi alligators). Hence there are not only causal pathways from DNA to phenotype, but also pathways that feed back from supragenetic resources (e.g. the cytoplasm) to the genetic material. Individual development can only be understood if multiple reciprocal ('coactional') influences, ranging from genes to epigenetic interactions and environmental influences, are taken into account. These multiple effects are exerted at and across a number of levels of organization. This implies that phenotypic properties are not derivable from genotypic properties in straightforward fashion. The role of emergence and self-organization in ontogeny is emphasized. Individual development is an historical process in which each new state (and function) of the developmental system builds upon previously existing states (and functions) of a causal, interactive network (Gottlieb, 1992, pp. 159–160). It is 'interactions, all the way down' (Elman et al., 1996, p. 319).

The notion of *parity* holds that neither genome nor epigenetic factors can be considered to have primacy over the other. Genes, according to developmentalism, are not in any sense *causally, informationally and explanatorily* privileged, as Crick's Central Dogma suggested. On the reductionist's picture, the information required for development is present in the molecular structure of the genes. The causal processes of development are initiated at this genetic level. Hence, ontogenetic processes can ultimately be accounted for in terms of DNA. These assumptions are questioned by developmentalism. As genes do not act alone, development is the result of *multiple causation* extending over a number of levels of organization. The unmistakable genetic influences are on a par with epigenetic modifications. There is no linear causal progression from genes to traits, but co-action (Gottlieb, 1992), that is, an inexorable intertwining of causes and their effects occurs in these developmental systems. According to developmentalism, it must be concluded that there is causal as well as explanatory parity.

In the context of investigations of the genome of the nematode *C. elegans*, Schaffner (1998) has drawn important conclusions with respect to the question of reductionism. He concludes that developmentalists are right that

even in *C. elegans* a tangle of causal factors is found and that preformationism must be wrong. Pleiotropy, plasticity and environmental context effects abound, and these properties prevent one-to-one mappings between genotype and phenotype from being established. These characteristics of development suggest there is *causal* parity (Schaffner, 1998). However, *explanatory and informational* parity must be denied, according to Schaffner. The genome is explanatorily and informationally priviliged. DNA has informational primacy, because this is where information flow in development starts. There is reason to see genes as representational, whereas the other causal factors involved in developmental processes cannot be claimed to carry information. This implies that genetic explanations, at least in some contexts, do seem to have a special status.

Heuristic Identification in Behavioral Genetics

Identifying Causes and Functions in Behavioral Genetics

Developmentalists may be right to insist that a multitude of causal factors is involved in bringing about a specific phenotypical consequence (see, e.g., Schaffner, 1998). However, the abundance of complexity and multiplicity in behavioral genetics does not preclude the possibility that there is something useful to genetic explanations. The identification of phenotypic traits with genetic properties (e.g. QTLs) may still be illuminating without succumbing to a simple-minded reductionism. The complex causal networks invoked by the developmentalists leave elbow room for the identification of causes ('genes for') with respect to a given causal background (Clark, 1998a, 1998b). In Clark's terminology, the genetic material functions as a primary *locus of plasticity*. Given a fixed ecological context, a change in the gene(s) would result in a change in the phenotypic trait. Holding constant the causal (i.e. the ecological and developmental) background, parts of the genome can be singled out as the factor that represents the trait on the basis of the fact (or conjecture) that it was the specific gene complex that was *selected* to replicate the phenotypic traits, that is, they have the *function* to replicate these traits.

It is in virtue of their having this function that, according to Clark's representational theory of genes, genes can still—with the lessons of developmentalism in mind—be seen as (weakly) representational: they code for traits, even though they are obviously not the only causally active factors involved. Although they are not complete sets of instructions (they rather set the parameters for development), genes are selected as encodings (i.e. representations) during evolution. Genes have this representational capacity relative to a normal environment and normal developmental processes. Against a stable backdrop of ecological and developmental conditions, a

gene or gene complex may be the principal difference-maker, the principal locus of plasticity. This constitutes the basis for genetic explanations in which phenotypic and genetic properties are identified. On this account, one is still allowed to speak of 'genes coding for traits', although this notion must be relativized to a set of normal ecological background conditions. Though genes may not be *causally* priviliged, they may be *explanatorily* prioritized in some contexts of investigation (see also Wheeler & Clark, 1999). They allow for genetic explanations that provide important means to isolate and pick out causal paths that are explanatorily salient, because they make a functional difference. Talking about 'genes for' constitutes a (context-relative) way to interpret what goes on in these labyrinthine, multiply constrained webs. Having thus rejected reductionism in genetics and developmental biology, we may now ask how to understand the identification of 'genes for'.

Limiting the HIT Rate

According to classical reductionism, identification was seen as the endpoint to which all science should be headed. Through identity statements, it was thought, the prime epistemic virtue of ontological economy could come to be honored (Causey, 1972). However, the identity theory is no longer in vogue. The so-called '*correlation objection*' against the identity theory states that identity statements are not entailed by correlation statements. This Hume-inspired objection is that on the basis of our observations of the world, insufficient evidence can be mounted to prove identity statements. They are 'metaphysical' in the sense that they cannot be empirically justified. McCauley and Bechtel (2001) answer this objection by pointing out that identification is a *means* or a *tool* towards greater integration of theories rather than a *goal* in science. Hypothesized identities, according to their heuristic identity theory (HIT), are important as a heuristic tool. Identity is employed not typically for any profound metaphysical reasons (i.e. ontological simplicity), but rather because it is opportune in the development of more detailed and more accurate explanations, models and theories (see also McCauley, 1981). With a twist on Leibniz's law of the identity of indiscernibles, McCauley and Bechtel (2001) argue that it is the *indiscernibility of identicals* that does most of the work. In the case of psychology and the neurosciences too, hypothesizing identity statements between mind and brain states will pay dividends. McCauley and Bechtel have illustrated this idea with the case of visual processing. What is known about the processing of visual information may effectively facilitate our search for the neural mechanisms of vision, and vice versa. Identificatory connections between neural and cognitive accounts of vision stimulate research on both sides of the identification. The application of Leibniz's inverted law to *interlevel* identities results in the *intralevel* modification of

concepts. These ideas on heuristic identification nicely dovetail with McCauley's (1996) account of the dynamical plurality of explanatory practices. HIT seems to capture what happens in behavioral genetics; it nicely describes the way in which identifications are hypothesized between genotypic and phenotypic levels without succumbing to naïve forms of genetic reductionism. At this point, it must be noted that McCauley and Bechtel's account of 'hypothetical identities' employs a notion of identity that is more liberal than what was intended in the classical understanding of reductionism. Here the ultimate goal was identification along the lines of the classical case in which the temperature of a gas is deemed identical to the mean kinetic energy of the molecules by which the gas is constituted. McCauley and Bechtel's example (see above) indicates that what they have in mind is the identification of function (i.e. visual perception of movement) and mechanisms (e.g. neural processing in the dorsal stream). We agree with this fairly tolerant interpretation of the notion of identity. In the case of behavioral genetics too, we submit, one may usefully speak of identities between functions and mechanisms, although these are not strict identities along the lines of Nagelian micro-reduction.

However, in our opinion, HIT still calls for some clarification. The proposal of identity statements may be a useful heuristic in multilevel theory development and the identification of genes. Nevertheless, we would like to warn against a wild proliferation of identity statements, in particular in the field of behavioral genetics. It is not true that every identification is as good as any other. Therefore, we would like to suggest some qualifications or restrictions on our use of the notion of heuristic identification. It will be argued that at least two constraints may be operative on such identifications: one concerning a preference for falsification over confirmation; the other concerning qualitative differences in cross-level identifications.

The virtues of misidentification. The first constraint is that one should distinguish between *falsification* and confirmation, between explanatory failure and success. The HIT rate should be limited by placing more emphasis on avoiding false positives. What we suggest is that identity claims, to cite William Wimsatt (1984), 'should be honored more in the breach than in the observance' (p. 498). Emphasis should be placed not so much on the explanatory and predictive successes as on the failures that identifications are capable of bringing to the fore. McCauley and Bechtel's choice of examples may betray an over-optimistic view of scientific progress; they are all success stories. Typically, identifications do not lead us on to a road of accumulating evidence towards complete confirmation of the hypothesized identity. Instead, falsification appears to play a more important role than verification. As Wimsatt (1984) says: 'Identity claims . . . provide probes of potentially unlimited sensitivity and depth for pinpointing sources of explanatory failures' (p. 498). It is not only successful identifications that

are important to science, but also failures and errors. Explanatory pluralism involves co-evolution, and co-evolution implies intralevel correction, and correction may follow from interlevel misidentification.

With an inflated HIT rate, we are left with the correlation objection all over again as we are stuck with too many false positives. Not just any established correlation should be elevated to the status of an identity statement. Such comparitive lack of attention to falsification seems at work in the 'gene of the week' phenomenon in behavioral genetics. Although identifications between phenotypic traits and genes or gene complexes abound, it appears that there is a relative lack of attention to falsifications. The 'gene of the week' phenomenon illustrates that genetic identity claims are wildly gyrating; useful insight and plausible conjecture are often hard to tell from wild surmise. In our view, this situation suggests that the HIT rate should be limited. Failing identifications direct our attention towards the source of the failure. By focusing on the relevant differences, one is able to improve on these identity statements.

The establishment of identificatory links between genes, neurotransmitters and personality traits may lead to change across all of these levels. For instance, it is now known that serotonine is involved in different aspects of what is known among psychologists as harm avoidance: depression, anxiety, hostility, and so on. This may lead, as Hamer and Copeland (1998, p. 103) speculate, to a reconfiguration of the upper-level concept. All of these aspects of harm avoidance may have in common that they involve the feeling of anger: towards oneself in the case of depression; towards others in the case of anger. Thus, these identificatory hypotheses provide a means to calibrate our psychological concepts against neurophysiological and genetic ones, and vice versa. The proposed identifications thus serve to coordinate multilevel inquiry. In the above example, our conceptions of personality traits may change under pressure of findings at a lower, in particular neurophysiological and genetic, level. Thus, *interlevel* explanatory failures may eventually be cashed in as *intralevel* theory changes, as Wimsatt realized when he stressed falsification of HITs.

Cross-level identifications. The second constraint is that we would like to focus attention on the fact that not all identifications are equally interesting. A salient feature of classical identity theory was its requirement that the relata in an identity claim (namely sense data and neurophysiological processes) should be independently accessed (Feigl, 1958). It appears that identifications that make a kind of *qualitative* leap are often more interesting and exciting than identifications that remain in the same qualitative domain. In particular, identifications between functions and causal mechanisms are often the most revealing and scientifically stimulating.

As argued above, explanatory failures point not only to gaps in the evidentiary chains, but in many cases to a deeper source as well. In fact, the

explanatory anomalies brought out by a particular identity statement testify to the distinctness of levels. Whereas incommensurability in *intralevel* contexts is tantamount to the elimination of one theory by the next one, incommensurability in *interlevel* contexts should be taken as evidence for the view that what we have in this particular case are two distinct levels of analysis. Incommensurability of theories, concepts and models is what you would expect in interlevel cases. Actually, through these hypothesized identity statements the assumption of distinct levels is further legitimized.

One particularly interesting case in which one encounters such qualitative leaps is the identification of a function with its underlying causal mechanisms. In multilevel analyses, the upper level is often a functionally characterized level. It specifies what a mechanism does or is supposed to do in a particular ecological context. Without functional perspectives, causally relevant explanations may not become available: functions single out what is causally relevant about the underlying (genetic and supragenetic) processes in the causal network. Mapping functions to loci on the genome (e.g. QTLs) is an important heuristic in furthering our knowledge of these networks. Such identificatory, genetic hypotheses provide useful instruments to localize causal mechanisms that fill the explanatory bill set by higher-level, functionally characterized phenomena. The most interesting cases are, we submit, those where the relata are described at qualitatively different levels, such as the leap from DNA to temperament (Hamer & Copeland, 1998).

Conclusions: Genetic Reductionism and Explanatory Pluralism

Genetic explanations, like other causal explanations, must be considered context-dependent (Schouten & Looren de Jong, 1996) and observer-relative. For certain questions, they have explanatory primacy (because they provide ways to trace out a trait's causal ancestry); for others, they have not. Further, a genetic factor is only relevant inasmuch as it refers to some stable ecological background. A genetic explanation presents a 'context-bound type identity' (Wimsatt, 1994, p. 228). It is 'a contextual generalization of uncertain but not too narrow scope, where the properties of the upper level thing are explained, ceteris paribus, by the operation of the spatio-temporally coincident lower level causal machine' (p. 229 n. 30). By picking out genes or QTLs, and connecting them to phenotypic traits, masses of other causal factors are ignored that may be important in other contexts. This implies that by offering these identifications one is deliberately simplifying matters. However, at the same time they do further our understanding of the processes involved in development (given such and such an ecological context). Therefore, these context-bound identifications must be considered *legitimate* simplifications.

Behavioral genetics invites an interpretation in terms of explanatory pluralism and heuristic identification. The new molecular behavioral genetics of which we have spoken can be interpreted in this light. Through new techniques such as QTL analysis, identificatory relations can be established between the molecular and phenotypic levels; more specifically, the levels of mind, brain and behavior. The warning must, however, be heeded that these types of research can easily result in overly ambitious, reductionistic interpretations. Developmentalism rightly insists that there are all kinds of qualifications to be made in interpreting genotype/phenotype relationships, as we have seen. Development is the product of complex causal interplay, even involving environmental factors. That said, however, this does not diminish the value of genetic explanations of the phenotype. On the contrary, it may still be possible to trace, through these tangled paths of causality, important factors that causally contribute to behavioral traits.

With the qualifications we have suggested, HIT promises to provide a plausible interpretation of the way in which research in behavioral genetics is actually carried out. It appears to be capable of describing how the genotypic and phenotypic levels are actually being (hypothetically) connected (and disconnected) in scientific practice. HIT's most notable virtue may be that it does so without yielding to genetic reductionism.

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